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DB=USPT,PGPB; PLUR=YES; OP=AND			
L3	recombinant adj (ndv or mumps or sendai or newcastle or simian or siv or syncytial)	32	L3
L2	recombinant near3 (ndv or mumps or sendai or newcastle or simian or siv or syncytial)	176	L2
L1	recombinant near3 (paramyxovir$ or morbillivirus or rubulavirus or pneumovirus)	17	L1

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☐ 1. 20030022376 . 27 Sep 01. 30 Jan 03. Paramyxovirus-derived RNP. Kitazato, Kaio, et al. 435/456; 435/235.1 C12N015/86 C12N007/00.

☐ 2. 20020197711 . 12 Jun 01. 26 Dec 02. Coxsackievirus B4 expression vectors and uses thereof. Ramsingh, Arlene I., et al. 435/320.1; 424/148.1 424/199.1 424/207.1 435/5 435/69.7 435/91.1 435/91.33 C12Q001/70 C12P021/04 C12P019/34 A61K039/42 A61K039/12 A61K039/21 C12N015/00 C12N015/09 C12N015/63 C12N015/70 C12N015/74.

☐ 3. 20020169306 . 27 Sep 01. 14 Nov 02. Envelope gene-deficient paramyxovirus vector. Kitazato, Kaio, et al. 536/23.4; 424/204.1 424/205.1 424/211.1 435/235.1 435/320.1 435/456 435/5 536/23.72 C12N015/86 C07H021/04 C12N007/00 C12Q001/70 A61K039/12 A61K039/155 C12N007/01 C12N015/00 C12N015/09 C12N015/63 C12N015/70 C12N015/74.

☐ 4. 20020155581 . 08 Dec 00. 24 Oct 02. Use of recombinant parainfluenza viruses (PIVs) as vectors to protect against infection and disease caused by PIV and other human pathogens. Murphy, Brian R., et al. 435/235.1; 424/211.1 424/212.1 424/434 435/320.1 536/23.72 C07H021/04 A61K039/165 C12N007/01 C12N015/09 C12N015/70 A61F013/00 A61K039/155 C12N015/00 C12N015/74 C12N007/00 C12N015/63.

☐ 5. 20020002143 . 30 Mar 01. 03 Jan 02. AIDS virus vaccines using sendai virus vector. Kano, Munehide, et al. 514/44; 435/320.1 A61K048/00 C12N015/867.

☐ 6. 6497873 . 22 Dec 98; 24 Dec 02. Recombinant Rhabdovirus containing a heterologous fusion protein. Whitt; Michael A., et al. 424/93.2; 435/235.1 435/239 435/320.1 435/440 435/455. A01N063/00 C12N007/00 C12N007/02 C12N015/00 C12N015/63.

☐ 7. 6426070 . 09 May 00; 30 Jul 02. Methods for inactivating enveloped RNA virus particles and compositions for use therewith. Rosenberg; Helene F., et al. 424/94.61; 424/185.1 424/211.1 435/238 435/69.2 530/350 530/380. A61K038/47 A61K039/155 A61K035/14 A61K038/16 G01N033/86.

☐ 8. 6156567 . 03 Jul 96; 05 Dec 00. Truncated transcriptionally active cytomegalovirus promoters. Fischer; Laurent. 435/325; 424/199.1 424/233.1 435/235.1 435/320.1 514/44 536/23.1 536/24.1. A61K039/235 C07H021/04 C07K014/075 C12N005/16.

☐ 9. 6130066 . 15 May 98; 10 Oct 00. Vectors having enhanced expression and methods of making and uses thereof. Tartaglia; James, et al. 435/69.1; 435/320.1 435/91.41 536/23.72. C12P021/06.

☐ 10. 6090393 . 03 Jul 96; 18 Jul 00. Recombinant canine adenoviruses, method for making and uses thereof. Fischer; Laurent. 424/233.1; 424/204.1 424/205.1 435/235.1. C12N007/00.

☐ 11. 6004777 . 12 Mar 97; 21 Dec 99. Vectors having enhanced expression, and methods of making and uses thereof. Tartaglia; James, et al. 435/69.1; 435/320.1 435/91.41 536/23.1 536/23.72. C12P021/00 C12N015/63 C12N015/66 C12N015/11.

☐ 12. 5990091 . 12 Mar 97; 23 Nov 99. Vectors having enhanced expression, and methods of making and uses thereof. Tartaglia; James, et al. 514/44; 424/93.2 435/320.1 435/69.1 435/91.4 435/91.41. C12N015/67 C12N015/86 A61K048/00.

☐ 13. 5911982 . 18 Apr 96; 15 Jun 99. Hz-1 virus persistence-associated-gene 1 (PAG1) promoter uses therefor, and compositions containing same or products therefrom. Chao; Yu-Chan. 424/93.2; 435/320.1 435/348 435/69.1 536/24.1. A61K031/70 C12P021/00 C12N005/06 C12N015/64.

☐ 14. 5759841 . 07 Jun 95; 02 Jun 98. Immunological composition of measles virus utilizing recombinant poxvirus. Paoletti; Enzo, et al. 435/235.1; 424/199.1 424/211.1 424/212.1 424/232.1 435/320.1 530/350. C12N007/00 C12N015/00 A61K039/12 A61K039/155.

☑ 15. 5503834 . 08 Jun 93; 02 Apr 96. Measles virus recombinant poxvirus vaccine. Paoletti; Enzo, et al. 424/199.1; 424/212.1 424/213.1 424/232.1 435/235.1 435/320.1. A61K039/165 A61K039/175 A61K039/285 C12N007/01.

☐ 16. 5288630 . 20 Nov 92; 22 Feb 94. Expression system for RSV glycoprotein F and G. Wathen; Michael W.. 435/348; 435/252.3 435/252.33 435/254.2 435/320.1 435/354 435/358 435/69.7 536/23.4. C12N005/10 C12N001/21 C12N001/19.

☐ 17. 5194595 . 20 Jun 90; 16 Mar 93. Chimeric glycoproteins containing immunogenic segments of the glycoproteins of human respiratory syncytial virus. Wathen; Michael W.. 530/395; 424/186.1 424/211.1 435/69.7. C07K013/00 C12N015/45 C12P021/02 A61K037/02.

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Terms	Documents
recombinant near3 (paramyxovir$ or morbillivirus or rubulavirus or pneumovirus)	17

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     (FILE 'HOME' ENTERED AT 11:54:33 ON 11 FEB 2003)

     FILE 'MEDLINE, CAPLUS, BIOSIS, SCISEARCH' ENTERED AT 11:54:47 ON 11 FEB
     2003
L1         31811 S PARAMYXOVIR? OR MORBILLIVIRUS OR RUBULAVIRUS OR PNEUMOVIRUS
L2         33969 S (MUMPS OR PARAINFLUENZA OR SENDAI OR MEASLES)(W)VIRUS
L3          2618 S (PHOCINE OR CANINE)(W)DISTEMPER(W)VIRUS
L4         78619 S (RESPIRATORY(W)SYNCYTIAL OR SIMIAN OR NEWCASTLE)(3A)VIRUS
L5        121083 S L1 OR L2 OR L3 OR L4
L6             3 S L5(8A)HETEROLOGOUS(W)(PROTEIN OR POLYPEPTIDE)
L7          2310 S L5(8A)(TUMOR(W)ANTIGEN OR CEA OR CARCINOEMBRYONIC(W)ANTIGEN
L8          1529 S L5(3A)(TUMOR(W)ANTIGEN OR CEA OR CARCINOEMBRYONIC(W)ANTIGEN
L9          1370 S L5(3A)(TUMOR(W)ANTIGEN OR CEA OR CARCINOEMBRYONIC(W)ANTIGEN
L10            2 DUP REM L6 (1 DUPLICATE REMOVED)
L11          593 DUP REM L9 (777 DUPLICATES REMOVED)
L12          224 S L5(3A)(ENCOD? OR EXPRESS?)(5A)(TUMOR(W)ANTIGEN OR CEA OR CAR
L13           94 DUP REM L12 (130 DUPLICATES REMOVED)
L14          100 S L5(3A)(ENCOD? OR EXPRESS?)(5A)(CEA OR CARCINOEMBRYONIC(W)ANT
L15           53 DUP REM L14 (47 DUPLICATES REMOVED)

=> d au ti so ab 20-53 l15

L15  ANSWER 20 OF 53  CAPLUS  COPYRIGHT 2003 ACS
IN   Kai, Chieko; Kato, Atsushi
TI   Preparation of cytokines using a Sendai virus
     expression system
SO   Eur. Pat. Appl., 13 pp.
     CODEN: EPXXDW
AB   The present invention provides a process for prepg. a cytokine
     inexpensively in large amts. by expressing the cytokine in a hen's egg
     using Sendai virus vector.  The cytokine obtained by the present process
     is expected to be useful as medication because it has sugar chains very
     similar to those of mammals.

L15  ANSWER 21 OF 53  CAPLUS  COPYRIGHT 2003 ACS
AU   Matsuse, Hiroto; Behera, Aruna K.; Kumar, Mukesh; Lockey, Richard F.;
     Mohapatra, Shyam S.
TI   Differential cytokine mRNA expression in Dermatophagoides farinae
     allergen-sensitized and respiratory syncytial virus-infected mice
SO   Microbes and Infection (2000), 2(7), 753-759
     CODEN: MCINFS; ISSN: 1286-4579
AB   The interaction between mite allergen sensitization and respiratory
     syncytial virus (RSV) infection at the level of cytokine mRNA expression
     was examd. in a murine model in the present study.  Primary RSV infection
     enhances expression of inflammatory cytokines such as IL-6, IFN-.gamma.,
     and eotaxin in the lung and upregulates the expression of Th2-like
     cytokines IL-10 and IL-13 in the spleen in BALB/c mice.  Mite
     antigen-sensitized and RSV-infected (ASRSV) mice show enhanced (P < 0.05)
     total serum IgE compared to antigen-sensitized mice.  However, the levels
     of viral mRNA in the lung tissues are comparable between RSV-infected and
     ASRSV mice.  It is concluded that compartmentalization of cytokine
     expression following RSV infection plays a role in the augmentation of
     Th2-like and IgE antibody response to RSV.

L15  ANSWER 22 OF 53  CAPLUS  COPYRIGHT 2003 ACS
AU   Wei, Lin; Dai, Jian-Xin; Sun, Shu-Han
TI   Construction and expression of coexpression plasmid of carcinoembryonic
     antigen and Newcastle disease virus HN gene
SO   Shengwu Gongcheng Xuebao (2000), 16(5), 641-644
     CODEN: SGXUED; ISSN: 1000-3061
AB   The eukaryotic expression plasmid (pcDNA3-CEA) contg. with the gene coding
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for the carcinoembryonic antigen (CEA) had been gotten with RT-PCR and gene recombination techniques. Using enzymolysis, ligation and other techniques, and eukaryotic coexpression plasmid (pcDNA3-CEA/HN) contg. 2 expression units of CEA and Newcastle disease virus HN gene that may have the function of immunoenhancement had been constructed. The plasmid will lay a foundation for further researching CEA nucleotide vaccine, adjuvant and their effect of special antitumor immune.

L15 ANSWER 23 OF 53 CAPLUS COPYRIGHT 2003 ACS
AU Gao, Jing; Choudhary, Suresh; Banerjee, Amiya K.; De, Bishnu P.
TI Human parainfluenza virus type 3 upregulates ICAM-1 (CD54) expression in a cytokine-independent manner
SO Gene Expression (2000), 9(3), 115-121
CODEN: GEEXEJ; ISSN: 1052-2166
AB Human parainfluenza virus type 3 (HPIV3) causes bronchiolitis, pneumonia, and croup in newborns and infants. Several studies have implicated intercellular adhesion mol.-1 (ICAM-1) in inflammation during infection by viruses. In this study, we investigated the potential for HPIV3 to induce ICAM-1 in HT1080 cells. FACS anal. showed that HPIV3 strongly induced ICAM-1 expression in these cells. The ICAM-1 induction was significantly reduced when the virions were UV inactivated prior to infection, indicating that ICAM-1 induction was mostly viral replication dependent. Culture supernatant of HPIV3-infected cells induced ICAM-1 at an extremely low level, indicating that virus-induced cytokines played only a minor role in the induction process. Consistent with this, potent inducers of ICAM-1 such as IFN-.gamma., TGF-.beta., and TNF-.alpha. were absent in the culture supernatant, but a significant amt. of IFN type I was present. By using U2A cells, which are defective in IFN type I signaling, we confirmed that ICAM-1 induction by HPIV3 occurred in a JAK/STAT signaling-independent manner. These data strongly indicate that HPIV3 induces ICAM-1 directly by viral antigens in a cytokine-independent manner; this induction may play a role in the inflammation during HPIV3 infection.

L15 ANSWER 24 OF 53 MEDLINE DUPLICATE 7
AU Tripp R A; Moore D; Jones L; Sullender W; Winter J; Anderson L J
TI Respiratory syncytial virus G and/or SH protein alters Th1 cytokines, natural killer cells, and neutrophils responding to pulmonary infection in BALB/c mice.
SO JOURNAL OF VIROLOGY, (1999 Sep) 73 (9) 7099-107.
Journal code: 0113724. ISSN: 0022-538X.
AB BALB/c mice sensitized to vaccinia virus **expressed** G protein of **respiratory syncytial virus** (RSV) develop a Th2-type **cytokine** response and pulmonary eosinophilia when challenged with live RSV. In this study, BALB/c mice were immunized or challenged with an RSV mutant lacking the G and SH proteins or with DNA vaccines coding for RSV G or F protein. F or G protein DNA vaccines were capable of sensitizing for pulmonary eosinophilia. The absence of the G and/or SH protein in the infecting virus resulted in a consistent increase both in pulmonary natural killer cells and in gamma interferon and tumor necrosis factor expression, as well as, with primary infection, a variable increase in neutrophils and CD11b(+) cells. The development of pulmonary eosinophilia in formalin-inactivated RSV-vaccinated mice required the presence of the G and/or SH protein in the challenge virus. These data show that G and/or SH protein has a marked impact on the inflammatory and innate immune response to RSV infection.

L15 ANSWER 25 OF 53 MEDLINE DUPLICATE 8
AU Deb S; Tessier C; Prigent-Tessier A; Barkai U; Ferguson-Gottschall S; Srivastava R K; Faliszek J; Gibori G
TI The expression of interleukin-6 (IL-6), IL-6 receptor, and gp130-kilodalton glycoprotein in the rat decidua and a decidual cell line: regulation by 17beta-estradiol and prolactin.
SO ENDOCRINOLOGY, (1999 Oct) 140 (10) 4442-50.
Journal code: 0375040. ISSN: 0013-7227.

AB The cytokine interleukin 6 (IL-6), a major mediator of immune and acute phase responses of the liver, has been implicated in the termination of pregnancy once expressed in the uterus. This study was undertaken to investigate the expression and regulation of genes encoding IL-6 and IL-6 receptor (IL-6R) in rat decidual tissue. Total RNA obtained from rat decidual tissue on different days of pseudopregnancy was analyzed by RT-PCR using specific primers for IL-6, IL-6R, and 130-kDa glycoprotein (gp130). Ribosomal L19 primers served as an internal control. IL-6R and gp130 were found to be expressed in the decidua throughout development, while no messenger RNA (mRNA) for IL-6 was detected. Interestingly, within several hours of culture, decidual explants acquired the ability to express IL-6. The apparent ability of decidual cells to express IL-6 and its lack of expression in vivo led us to examine whether the IL-6 gene is actively inhibited. Primary decidual cells were cultured in the presence of estradiol, progesterone, or PRL. Progesterone showed no effect, whereas estradiol and PRL reduced the level of IL-6 mRNA expression. To examine the mechanism by which these hormones inhibit **IL**-6 **expression**, we used a **simian virus** 40-transformed decidual cell line (GG-AD), which expresses only estrogen receptor-beta (ERbeta). Like primary decidual cells in culture, GG-AD cells express IL-6, IL-6R, and gp130 mRNA. When cultured in the presence of estradiol (0-100 ng/ml), mRNA for IL-6 and its receptor components were down-regulated in a dose-dependent manner. Estradiol also caused a dose-dependent decrease in IL-6 protein secretion into the culture medium. The inhibitory effect of estradiol on IL-6 mRNA expression was reversed by the antiestrogen ICI-164,384. Similar inhibition of IL-6 and gp130 mRNA expression was observed with PRL treatment. However, PRL had no effect on IL-6R mRNA levels. PRL inhibition of IL-6 expression was totally reversed by tyrphostin AG490, a JAK2 inhibitor. In summary, the results of this investigation indicate that IL-6 expression, which is detrimental to the maintenance of pregnancy, is inhibited in the rat decidual tissue. This inhibition is induced by PRL and estradiol, which down-regulate not only IL-6 expression, but also the expression of IL-6 receptor and signaling proteins. The results also suggest that PRL signaling to the IL-6 gene is mediated through the long form of PRL receptor and involves JAK2 activation, whereas that of estradiol can be transduced by estrogen receptor-beta.

L15 ANSWER 26 OF 53 MEDLINE DUPLICATE 9
AU Dmoszynska A; Kandefer-Szerszen M; Rolinski J; Legiec W; Kaminska T
TI Influence of low dose rIL-2 treatment on endogenous cytokine production, expression of surface IL-2R and the level of soluble IL-2R in patients with minimal residual disease.
SO LEUKEMIA AND LYMPHOMA, (1999 Oct) 35 (3-4) 355-66.
Journal code: 9007422. ISSN: 1042-8194.
AB This study was designed to investigate the immunomodulatory effect of low-dose IL-2 therapy (100 microg/day for 3 weeks) on interferon (IFN), tumor necrosis factor (TNF) production in vivo and in vitro and on the expression of IL-2Ralpha/beta and soluble form of IL-2Ralpha. Patients enrolled in the study suffered from multiple myeloma (MM), Hodgkin's disease (HD) and non-Hodgkin's lymphoma (NHL) All of them were in remission after chemotherapy or radiotherapy. Our results indicated that IL-2 given subcutaneously at a low dose of 100 microg/day for 3 weeks induced IFN-gamma and TNF-alpha in plasma (measured 24 hrs after the last dose of IL-2) and affected the ability of blood leukocytes to produce cytokines. Production of IFN-gamma induced in vitro with PHA was enhanced, but TNF-alpha production induced by lipopolysaccharide (LPS) and virus (**Newcastle** Disease **Virus**) was depressed. The **expression** of both: surface **IL**-2R, especially beta subunit on total population of lymphocytes and NK cells, and soluble form of IL-2R, of chain were significantly enhanced after low-dose IL-2 therapy. Low dose IL-2 therapy was well tolerated by all patients, and side effects not exceeding II grade of toxicity according to WHO scale were observed. Five patients with MM relapsed 3-10 month after cessation

of IL-2 therapy, but all patients with Hodgkin's and non-Hodgkin's lymphomas are still in remission (20 months of observation).

L15 ANSWER 27 OF 53 MEDLINE DUPLICATE 10
AU Frisk A L; Baumgartner W; Grone A
TI Dominating interleukin-10 mRNA expression induction in cerebrospinal fluid cells of dogs with natural canine distemper virus induced demyelinating and non-demyelinating CNS lesions.
SO JOURNAL OF NEUROIMMUNOLOGY, (1999 Jun 1) 97 (1-2) 102-9.
Journal code: 8109498. ISSN: 0165-5728.
AB Canine distemper virus (CDV) infection in dogs is commonly associated with demyelinating leukoencephalitis (DL). Although the mechanism of primary demyelination in distemper remains undetermined recent studies showed a direct virus-induced cytolysis in early non-inflammatory and immune-mediated mechanisms in inflammatory lesions. To further investigate the pathogenesis of this **morbillivirus**-induced demyelination the **expression** of a variety of **cytokine** mRNA species (interleukin (IL)-1beta, IL-2, IL-6, IL-10, IL-12, tumor necrosis factor (TNF)-alpha, transforming growth factor (TGF)-beta1, and interferon (IFN)-gamma in cerebrospinal fluid cells of 12 dogs with CDV encephalitis was investigated employing reverse transcription-polymerase chain reaction (RT-PCR) and these findings were correlated to the type of CNS lesions. Neuropathology revealed the whole spectrum of distemper DL lesions from acute to chronic alterations, however, most plaques lacked active demyelination. Three control animals were devoid of any cytokine expression, whereas in distemper animals IL-10 transcripts were found in nine dogs with acute and chronic lesions. IL-6, TNF, and TGF mRNA was found in six, four, and three animals, respectively. IL-12 and IFN-gamma, suggestive of a TH1-like dominated immune response, were detected only in one animal with chronic lesions. Summarized, TNF and IL-6, associated with disease exacerbation, and IL-10 and TGF, indicative of remission, were often observed simultaneously in distemper DL and could not be assigned to a specific disease stage. However IL-10 mRNA remained the most frequently detected cytokine indicating a stage of inactivity in most animals investigated.

L15 ANSWER 28 OF 53 MEDLINE DUPLICATE 11
AU Singh M; Billeter M A
TI A recombinant measles virus expressing biologically active human interleukin-12.
SO JOURNAL OF GENERAL VIROLOGY, (1999 Jan) 80 (Pt 1) 101-6.
Journal code: 0077340. ISSN: 0022-1317.
AB Suppression of cell-mediated immunity (CMI) is well-documented during and after measles. This immunosuppression is suggested to result from decreased production of interleukin-12 (IL-12), a key interleukin for CMI. In an attempt to clearly discern the role of IL-12 in measles-induced immunosuppression, a **measles virus** (MV) that **expresses** biologically active human **IL**-12 was generated. This was achieved by inserting the coding sequences of the two subunits (p35 and p40) of human IL-12 separated by an internal ribosome entry site in an additional transcription unit between the H and the L genes of MV. Although the IL-12-expressing MV grew slightly slower than the normal MV, it stably maintained the inserted sequences (3.2 kb) and uniformly expressed the foreign genes after 10 passages in cell culture. These findings suggest that MV is a well-suited vector for delivery of proteins of immunogenic and therapeutic importance.

L15 ANSWER 29 OF 53 CAPLUS COPYRIGHT 2003 ACS
IN Bennett, Alice Marie
TI Simian herpes b virus glycoprotein d and its application in engineering recombinant viral vaccines for humoral immunostimulation
SO Brit. UK Pat. Appl., 23 pp.
CODEN: BAXXDU
AB A prophylactic or therapeutic vaccine for use in protecting mammals such

as humans or animals is described. The vaccine is based upon the glycoprotein D (gD) of simian herpes B virus. Specifically, the vaccine comprises gD of B virus or a fragment or variant which is capable of producing a protective immune response in a mammal to which it is administered is presented. Genetic vectors including nucleotide sequences encoding glycoprotein d or fragments or variants is described.

L15 ANSWER 30 OF 53 CAPLUS COPYRIGHT 2003 ACS
AU McInnes, E.; Collins, R. A.; Taylor, G.
TI Cytokine expression in pulmonary and peripheral blood mononuclear cells from calves infected with bovine respiratory syncytial virus
SO Research in Veterinary Science (1998), 64(2), 163-166
CODEN: RVTSA9; ISSN: 0034-5288
AB The possible involvement of cytokines in the acute viral pneumonia induced by respiratory syncytial virus (RSV) infection was studied in calves. The pattern of cytokine mRNA expression in mononuclear cells (MNC) isolated from the lung and peripheral blood of six gnotobiotic calves infected seven days previously with bovine RSV were analyzed by reverse-transcription polymerase chain reaction using primers specific for bovine cytokines. The pattern of cytokines detected indicated a mixed type of response to RSV infection as mRNAs for IFN-.gamma., IL-2, IL-4 and IL-10 were detected in pulmonary and peripheral blood MNC from calves with extensive pneumonic consolidation. In contrast, only mRNA for IFN-.gamma. was detected in MNC from the lungs and peripheral blood of uninfected animals. These data provide preliminary information on the potential range of cytokines produced in calves following infection with bovine RS virus.

L15 ANSWER 31 OF 53 CAPLUS COPYRIGHT 2003 ACS
AU Smit-McBride, Zeljka; Mattapallil, Joseph J.; Villinger, Francois; Ansari, Aftab A.; Dandekar, Satya
TI Intracellular cytokine expression in the CD4+ and CD8+ T cells from intestinal mucosa of simian immunodeficiency virus infected macaques
SO Journal of Medical Primatology (1998), 27(2/3), 129-140
CODEN: JMPMAO; ISSN: 0047-2565
AB Isolated intraepithelial lymphocytes (IEL) and lamina propria lymphocytes (LPL) from jejunum of SIV infected animals were examd. for alterations in basal cytokine expression by RT-PCR. Remarkable changes in IFN.gamma. and IL-10 RNA levels were obsd. in IEL and LPL in SIV infection while IL-4 and IL-2 RNA levels remained unaltered. In addn., the CD4+ and CD8+ LPL were examd. for intracellular cytokine prodn. following mitogenic activation by flow cytometry. Both CD4+ and CD8+ T lymphocytes in intestinal mucosa retained the potential to produce IFN.gamma. in response to mitogenic stimulation in vitro, without a remarkable change in IL-4 prodn. The dominant IFN.gamma. cytokine response could be one of the major contributing factors in SIV assocd. enteropathy.

L15 ANSWER 32 OF 53 CAPLUS COPYRIGHT 2003 ACS
AU Kawabata, Shigetada; Miller, Christopher J.; Lehner, Thomas; Fujhashi, Kohtaro; Kubota, Mitsuru; McGhee, Jerry R.; Imaoka, Koichi; Hiroi, Takachika; Kiyono, Hiroshi
TI Induction of Th2 cytokine expression for p27-specific IgA B cell responses after targeted lymph node immunization with simian immunodeficiency virus antigens in rhesus macaques
SO Journal of Infectious Diseases (1998), 177(1), 26-33
CODEN: JIDIAQ; ISSN: 0022-1899
AB To det. if there is an assocn. between the isotype of simian immunodeficiency virus (SIV)-specific B cell responses and the profile of Th1 and Th2 cytokine expression, rhesus macaques were immunized with SIV antigens via the iliac lymph nodes, using a targeted lymph node (TLN) immunization procedure. When CD4+ T cells purified from antigen-stimulated peripheral blood mononuclear cells were analyzed, the levels of Th2 cytokine prodn. were gradually increased after the second and third immunizations. However, interferon-.gamma. prodn. did not

change. Anal. of SIV-specific B cell responses revealed that the main isotype was IgG after the second and third immunizations. In addn., a peak of SIV-specific IgA B cell responses was noted following the third immunization. These findings suggest that the induction of Th2 type responses in TLN-immunized rhesus macaques reflects the sequence of initial induction of SIV-specific IgG-producing cells followed by IgA-secreting cells.

L15 ANSWER 33 OF 53 CAPLUS COPYRIGHT 2003 ACS
AU Grone, A.; Frisk, A. L.; Baumgartner, W.
TI Cytokine mRNA expression in whole blood samples from dogs with natural canine distemper virus infection
SO Veterinary Immunology and Immunopathology (1998), 65(1), 11-28
CODEN: VIIMDS; ISSN: 0165-2427
AB Cytokines are sol. polypeptides with many physiol. functions and a special role during infection and inflammation. Little is known about cytokine regulation in naturally occurring viral diseases of animals. Esp. the role of cytokines in the development and progression of lesions in canine distemper virus (CDV) infection in dogs is largely unknown. Whole blood samples from 14 dogs with CDV infection and three dogs suffering from non-distemper diseases were examd. for mRNA of pro-inflammatory cytokines such as interleukin-I.beta. (IL-1), interleukin-6 (IL-6), interleukin-8 (IL-8), interleukin-12 (IL-12), tumor necrosis factor-.alpha. (TNF), interferon-.gamma. (IFN), and the anti-inflammatory transforming growth factor-.beta.1 (TGF) using reverse transcription polymerase chain reaction (RT-PCR). Blood samples from the three dogs that showed no clin. abnormalities during a pre-vaccination phys. examn. served as control. CDV infection was confirmed by post-mortem immunohistochem. for CDV nucleoprotein. The degree of immunoreactivity and the no. of virus antigen pos. organs were expressed as antigen index. IFN transcripts were not identified in any dog and IL-8 transcripts were present in RNA isolates from all 20 dogs. None of the other cytokines was detected in control animals. IL-1 and IL-6 were each found in one non-distemper dog and TGF transcripts were amplified in two dogs with non-distemper disease. The following transcripts were found in variable nos. in distemper dogs: IL-1 (7/14 dogs), IL-6 (3/14 dogs), IL-12 (3/14 dogs), TNF (8/14 dogs), and TGF (10/14 dogs) with multiple cytokines in ten dogs. No cytokine transcripts were detected in three distemper dogs. There was no obvious correlation between cytokine mRNA expression and respiratory and gastrointestinal tract diseases. In the CNS, demyelination was frequently assocd. with IL-1, IL-12, TNF and TGF mRNA expression. IL-6 transcripts were found only in animals with early CNS lesions and TGF was the only detectable cytokine in an animal with chronic demyelination. Lack of detectable cytokine transcripts in whole blood samples was assocd. with a high antigen index and viremia, indicating that an overwhelming virus infection may suppress cytokine prodn., possibly due to paralysis of the immune system. Simultaneous occurrence of pro- and anti-inflammatory cytokines in whole blood prepn. from most of the dogs with distemper, indicated a complex most likely disease stage dependent orchestrated cytokine expression.

L15 ANSWER 34 OF 53 CAPLUS COPYRIGHT 2003 ACS
IN Collins, Peter L.
TI In vivo and vitro packaging of infectious respiratory syncytial virus using cloned viral nucleic acids
SO PCT Int. Appl., 65 pp.
CODEN: PIXXD2
AB Methods of packaging mammalian respiratory syncytial virus (RSV) antigenomes in vivo or in a cell-free system for the prepn. of virus for therapeutic use, esp. vaccines, are described. The methods use cloned genes for the proteins involved in the minimal infectious form of the virus: nucleocapsid (N), nucleocapsid phosphoprotein (P), large (L) polymerase protein, and an RNA polymerase elongation factor, such as RSV M2, and a transcription unit encoding the antigenome to manuf. infectious

virus. The methods are applicable to human, bovine or murine RSV or RSV-like viruses, and variants carrying foreign genes or parts of several different RSV genomes. A modified RSV antigenome can be used to produce desired phenotypic changes, such as attenuated viruses for vaccine use. A cDNA for a viral antigenome, including a terminal ribozyme to ensure accurate cleavage of a primary transcription product was constructed. This was introduced, along with expression constructs for the G, F, L, and M2 genes, into HEp-2 cells with the formation of infectious virus particles. Optimization expts. showed that the RNA polymerase elongation factor was crit. to successful formation of infectious particles. The prepn. of RSV with altered phenotypes, e.g. cold-sensitivity, or carrying a foreign gene was also demonstrated.

L15 ANSWER 35 OF 53 CAPLUS COPYRIGHT 2003 ACS
AU Waris, Matti E.; Tsou, Cecilia; Erdman, Dean D.; Day, Diane B.; Anderson, Larry J.
TI Priming with live respiratory syncytial virus (RSV) prevents the enhanced pulmonary inflammatory response seen after RSV challenge in BALB/c mice immunized with formalin-inactivated RSV
SO Journal of Virology (1997), 71(9), 6935-6939
CODEN: JOVIAM; ISSN: 0022-538X
AB To investigate enhanced disease assocd. with a formalin-inactivated (FI) respiratory syncytial virus (RSV) vaccine, we studied the pulmonary inflammatory response to RSV in BALB/c mice immunized with live RSV, FI-RSV, or combinations of the two. After RSV challenge, the no. of granular cells, the ratio of CD4+/CD8+ lymphocytes, and the level of Th2-like cytokine mRNAs in the bronchoalveolar lavage specimens in mice immunized first with live RSV and then with FI-RSV were lower than that in FI-RSV-immunized mice and close to that in live RSV-immunized mice. These data suggest that prior live RSV infection prevents most of the enhanced inflammatory response seen in FI-RSV-immunized mice and might explain lack of enhanced disease in older FI-RSV-immunized children. A live RSV vaccine might similarly decrease the risk of enhanced disease with non-live RSV vaccines.

L15 ANSWER 36 OF 53 MEDLINE DUPLICATE 12
AU Srikiatkhachorn A; Braciale T J
TI Virus-specific memory and effector T lymphocytes exhibit different cytokine responses to antigens during experimental murine respiratory syncytial virus infection.
SO JOURNAL OF VIROLOGY, (1997 Jan) 71 (1) 678-85.
Journal code: 0113724. ISSN: 0022-538X.
AB Mice sensitized to the G (attachment) or F (fusion) glycoproteins of **respiratory syncytial virus** (RSV) **expressed** different patterns of **cytokine** production and lung pathology when challenged by intranasal infection with RSV. Five days after challenge, mice sensitized to G glycoprotein produced high levels of interleukin-4 (IL-4) and IL-5 in the lungs and spleens and developed extensive pulmonary eosinophilia, while mice sensitized to F glycoprotein produced IL-2 and developed a mononuclear cell infiltration. Memory lymphocytes isolated 2 weeks after intranasal challenge of mice primed to the G or F glycoprotein secreted only IL-2 and gamma interferon (IFN-gamma) when stimulated with RSV. IL-4 and IL-5 production characteristic of Th2-type effectors in the lung was observed only after multiple rounds of in vitro stimulation of RSV G-specific memory T lymphocytes with antigen. Also IFN-gamma production appeared to play only a minor role in the expression of pulmonary pathology characteristic of Th1 or Th2 T-lymphocyte responses, because mice genetically deficient in IFN-gamma production by gene disruption displayed the same pattern of pulmonary inflammation to RSV infection after priming to RSV F or G as conventional mice. These results suggest that effector T lymphocytes exhibit a different pattern of cytokine production than memory T-lymphocyte precursors precommitted to a Th1 or Th2 pattern of differentiation. Furthermore, these observations raise the possibility

that the cytokine response of human memory T lymphocytes after a single exposure to antigen in vitro may not accurately reflect the cytokine response of differentiated effector T lymphocytes at the site of infection in vivo.

L15 ANSWER 37 OF 53 CAPLUS COPYRIGHT 2003 ACS
AU Stebbings, Richard J.; Lines, Jenny L.; Almond, Neil M.; Stott, Edward J.; Walker, Kenneth B.
TI Cytokine profiling of simian immunodeficiency virus infection of cynomolgus macaques. Pathogenic SIVmac251 pJ5C versus non-pathogenic attenuated SIVmac251 pC8C reveals divergence
SO Biochemical Society Transactions (1997), 25(2), 285S
CODEN: BCSTB5; ISSN: 0300-5127
AB The non-pathogenic SIV clone SIVmac251 pC8C is attenuated by a 12 bp deletion in the nef ORF>. Vaccination of macaques with this partly nef deleted SIV mutant results in a low level viremia that protects against superinfection with the pathogenic clone pJ5C. The mechanism of this superinfection resistance and the loss of pathogenicity in nef-deleted SIV is as yet unexplored. Here, in order to gain insights into this model of protection, the authors compared the cytokine profiles of four macaques infected with pathogenic J5C with four animals infected with non-pathogenic C*C virus.

L15 ANSWER 38 OF 53 MEDLINE DUPLICATE 13
AU Benveniste O; Vaslin B; Le Grand R; Cheret A; Matheux F; Theodoro F; Cranage M P; Dormont D
TI Comparative interleukin (IL-2)/interferon IFN-gamma and IL-4/IL-10 responses during acute infection of macaques inoculated with attenuated nef-truncated or pathogenic SICmac251 virus.
SO PROCEEDINGS OF THE NATIONAL ACADEMY OF SCIENCES OF THE UNITED STATES OF AMERICA, (1996 Apr 16) 93 (8) 3658-63.
Journal code: 7505876. ISSN: 0027-8424.
AB Comparison of immune responses to infection by a pathogenic or a nonpathogenic immunodeficiency virus in macaques may provide insights into pathogenetic events leading to simian AIDS. This work is aimed at exploring **cytokine expression** during infection by **simian** immunodeficiency **virus** (SIV). We used semiquantitative reverse transcription-PCR to monitor interleukin (IL)-2/interferon (IFN)-gamma (Th1-like), and IL-4/IL-10 (Th2-like) expression in unmanipulated peripheral blood mononuclear cells (PBMCs), during the acute phase of infection of eight cynomolgus macaques (Macaca fascicularis) with a pathogenic primary isolate of SIVmac251 (full-length nef), and of four other cynomolgus macaques by an attenuated molecular clone of SIVmac251 (nef-truncated). All the monkeys became infected, as clearly shown by the presence of infected PBMCs and by seroconversion. Nevertheless, PBMC-associated virus loads and p27 antigenemia in monkeys infected by the attenuated virus clone remained lower than those observed in animals infected with the pathogenic SIVmac251 isolate. A rise of IL-10 mRNA expression occurred in both groups of monkeys coincident with the peak of viral replication. In monkeys infected with the pathogenic SIVmac251, IL-2, IL-4, and IFN-gamma mRNAs were either weakly detectable or undetectable. On the contrary, animals infected by the attenuated virus exhibited an overexpression of these cytokine mRNAs during the first weeks after inoculation. The lack of expression of these cytokines in monkeys infected with the pathogenic primary isolate may reflect early immunodeficiency.

L15 ANSWER 39 OF 53 MEDLINE DUPLICATE 14
AU Tsutsumi H; Matsuda K; Sone S; Takeuchi R; Chiba S
TI Respiratory syncytial virus-induced cytokine production by neonatal macrophages.
SO CLINICAL AND EXPERIMENTAL IMMUNOLOGY, (1996 Dec) 106 (3) 442-6.
Journal code: 0057202. ISSN: 0009-9104.
AB The induction of immunoregulatory cytokines IL-1 beta, IL-6, IL-12, tumour

necrosis factor-alpha (TNF-alpha) and interferon-gamma (IFN-gamma) was studied with neonatal (cord blood) monocyte-derived macrophages (MDM) after in vitro infection with **respiratory syncytial virus** (RSV). The **expression** of mRNAs for these **cytokines** in RSV-infected MDM was examined by reverse transcriptase-polymerase chain reaction (RT-PCR). The activities of these cytokines were assayed by ELISA. Significant increase of expression of mRNA for IL-6, IL-12, TNF-alpha and IFN-gamma occurred within 2 h after infection and decreased within 6 h after infection. At 20 h after infection the MDM produced and secreted moderate levels of IL-6 and TNF-alpha; however, no IL-12 and IFN-gamma activities were detected. Moderate IL-1 beta mRNA was expressed before RSV infection, and its expression increased at 2 h after infection. However, no detectable IL-1 beta was secreted in culture fluids. These observations suggest that RSV-infected neonatal macrophages produce and secrete IL-6 and TNF-alpha quickly during the eclipse phase of RSV infection and therefore may play a prominent role in the initiation of the immune response to RSV.

L15 ANSWER 40 OF 53 MEDLINE DUPLICATE 15
AU Mastronarde J G; He B; Monick M M; Mukaida N; Matsushima K; Hunninghake G W
TI Induction of interleukin (**IL**)-8 gene **expression** by **respiratory syncytial virus** involves activation of nuclear factor (NF)-kappa B and NF-IL-6.
SO JOURNAL OF INFECTIOUS DISEASES, (1996 Aug) 174 (2) 262-7.
Journal code: 0413675. ISSN: 0022-1899.
AB Respiratory syncytial virus (RSV) preferentially infects respiratory epithelium and is an important cause of lower respiratory tract infections in young children. RSV induces the production of interleukin (IL)-8 in airway epithelial cells; however, the mechanism of this induction is not known. To define the mechanism by which RSV induces IL-8 gene activation, A549 epithelial cells were transfected with plasmids containing serial deletions of the 5'-flanking region of the IL-8 gene and then exposed to RSV for 24 h. A positive cooperative effect of the binding sites for the transcription factors, nuclear factor (NF)-kappa B and NF-IL-6, was observed. Mutations in either region abates responsiveness of the promoter to RSV infection. RSV also increases activation of the NF-kappa B and NF-IL-6 transcription factors. These data suggest that RSV may increase IL-8 production in airway epithelium partly via activation of the transcription factors NF-kappa B and NF-IL-6.

L15 ANSWER 41 OF 53 MEDLINE DUPLICATE 16
AU Megyeri K; Au W C; Rosztoczy I; Raj N B; Miller R L; Tomai M A; Pitha P M
TI Stimulation of interferon and cytokine gene expression by imiquimod and stimulation by Sendai virus utilize similar signal transduction pathways.
SO MOLECULAR AND CELLULAR BIOLOGY, (1995 Apr) 15 (4) 2207-18.
Journal code: 8109087. ISSN: 0270-7306.
AB The imidazoquinolineamine derivative 1-(2-methyl propyl)-1H-imidazole [4,5-c]quinoline-4-amine (imiquimod) has been shown to induce alpha interferon (IFN-alpha) synthesis both in vivo and in peripheral blood mononuclear cells in vitro. In this study, we show that, in these cells, imiquimod induces expression of several IFNA genes (IFNA1, IFNA2, IFNA5, IFNA6, and IFNA8) as well as the IFNB gene. Imiquimod also induced the expression of interleukin (IL)-6, IL-8, and tumor necrosis factor alpha genes. Expression of all these genes was transient, independent of cellular protein synthesis, and inhibited in the presence of tyrosine kinase and protein kinase C inhibitors. Infection with **Sendai virus** led to **expression** of a similar set of **cytokine** genes and several of the IFNA genes. Imiquimod stimulates binding of several induction-specific nuclear complexes: (i) the NF-kappa B-specific complexes binding to the kappa B enhancer present in the promoters of all cytokine genes, but not in IFNA genes, and (ii) the complex(es) binding to the A4F1 site, 5'-GTAAAGAAAGT-3', conserved in the inducible element of IFNA genes. These results indicate that imiquimod,

similar to viral infection, stimulates expression of a large number of cytokine genes, including IFN-alpha/beta, and that the signal transduction pathway induced by both of these stimuli requires tyrosine kinase and protein kinase activity.

L15 ANSWER 42 OF 53 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC.
AU Luciw, Paul A. (1); Low, Tessie (1); Stout, Mike (1); Ansari, Aftab; Villinger, Francois
TI Replication-competent **simian** immunodeficiency **virus** (SIV) vectors for **expression** of **cytokine** genes: Implications for live-attenuated viral vaccines.
SO Journal of Medical Primatology, (1995) Vol. 24, No. 4, pp. 183. Meeting Info.: 13th Annual Symposium on Nonhuman Primate Models for AIDS Monterey, California, USA November 5-8, 1994 ISSN: 0047-2565.

L15 ANSWER 43 OF 53 CAPLUS COPYRIGHT 2003 ACS
AU Tang, Yi-Wei; Graham, Barney S.
TI Anti-IL-4 treatment at immunization modulates cytokine expression, reduces illness, and increases cytotoxic T lymphocyte activity in mice challenged with respiratory syncytial virus
SO Journal of Clinical Investigation (1994), 94(5), 1953-8 CODEN: JCINAO; ISSN: 0021-9738
AB Upon respiratory syncytial virus (RSV) challenge, mice previously immunized i.m. with inactivated whole virus express a Th2-like pattern of cytokine mRNA, while mice immunized with liver virus intranasally express a Th1-like pattern. In this study, we evaluated the effects of anti-IL-4 treatment on the induction of immune responses after immunization. Mice treated with anti-IL-4 at the time of immunization with inactivated RSV had reduced clin. illness after live virus challenge, as measured by wt. loss, illness score, and virus replication. This was assocd. with an augmented CD8+ cytotoxic T lymphocyte (CTL) activity, increased expression of IFN-.gamma. mRNA relative to IL-4 mRNA, and a higher titer of RSV-specific IgG2a in the anti-IL-4 treated mice before challenge. Anti-IL-4 administration at the time of challenge had no effects on illness, Ig isotype, or cytokine patterns. These results suggest that inhibition of IL-4 action at immunization can shift the selective activation of lymphocytes to a more Th1-like response. This cytokine milieu is assocd. with augmented CTL activity, which may be the factor responsible for rapid viral clearance and reduced illness at the time of remote RSV challenge.

L15 ANSWER 44 OF 53 CAPLUS COPYRIGHT 2003 ACS
AU Khatissian, E.; Chakrabarti, L.; Hurtrel, B.
TI Cytokine patterns in lymph nodes during the early stages of simian immunodeficiency virus infection
SO Research in Immunology (1994), 145(8-9), 702-5 CODEN: RIMME5; ISSN: 0923-2494
AB In this report, the authors examd. the range of cytokines produced by activated macrophages and Th1 or Th2 cells as quantified using reverse transcriptase PCR.

L15 ANSWER 45 OF 53 CAPLUS COPYRIGHT 2003 ACS
AU Yamabe, Toshio; Dhir, Gita; Cowan, Elliot P.; Wolf, Aizik L.; Bergey, Gregory K.; Krumholz, Allan; Barry, Elizabeth; Hoffman, Paul M.; Dhib-Jalbut, Suhayl
TI Cytokine-gene expression in measles-infected adult human glial cells
SO Journal of Neuroimmunology (1994), 49(1-2), 171-9 CODEN: JNRIDW; ISSN: 0165-5728
AB The expression of interleukin (IL)-1.beta., IL-6 and tumor necrosis factor (TNF) .alpha. transcripts in cultured human glial cells was examd. using reverse transcription followed by polymerase chain reaction (PCR) amplification and Southern blot quantitation. Microglial cultures derived from brain biopsy specimens from three different individuals expressed

transcripts for the three cytokines under basal culture conditions. This expression was enhanced in response to measles virus (MV) infection (IL-1.beta., 2.2-8.8-fold; IL-6, 2.5-8.4-fold; TNF.alpha., 2.2-3.2-fold). Neither IL-1.beta. nor TNF.alpha. transcripts were detectable in undissociated brain tissue from two individuals, suggesting that the basal expression of these cytokines in culture may have been induced by tissue dissocn. or by the culture conditions. Oligodendrocytes did not express cytokine transcripts under basal culture conditions, and IL-1.beta. and IL-6 but not TNF.alpha. transcripts could be induced by MV. Similarly, meningeal fibroblasts expressed IL-1.beta. and IL-6 but not TNF.alpha. in response to MV-infection, suggesting that the prodn. of TNF.alpha. is more cell type-restricted than either IL-1.beta. or IL-6. Thus, adult human microglia can participate in the inflammatory response to MV infection in the CNS by producing cytokines that contribute to inflammation and demyelination. In addn., besides their role in myelination, oligodendrocytes can potentially influence immunoreactivity in the CNS by producing IL-1.beta. and IL-6.

L15 ANSWER 46 OF 53 CAPLUS COPYRIGHT 2003 ACS
AU Graham, Barney S.; Henderson, Gregory S.; Tang, Yi Wei; Lu, Xiaotao; Neuzil, Kathleen M.; Colley, Daniel G.
TI Priming immunization determines T helper cytokine mRNA expression patterns in lungs of mice challenged with respiratory syncytial virus
SO Journal of Immunology (1993), 151(4), 2032-40
CODEN: JOIMA3; ISSN: 0022-1767
AB Defining the mechanism for the vaccine-enhanced illness assocd. with respiratory syncytial virus (RSV) is crit. for advancing RSV vaccine development. Previous studies in which infants were vaccinated with formalin-inactivated alum-pptd. whole virus did not protect from RSV infection, and those infected had a high incidence of severe illness. In contrast, previous clin. trials evaluating live attenuated RSV showed no assocd. vaccine-enhanced illness. Here, a mouse model was used to explore the immunopathogenesis of RSV infection. In this study cytokine mRNA expression was examd. using 32P-labeled oligonucleotide probes in Northern blot analyses of polyA RNA extd. from lungs of mice primed with various vaccine prepns. then challenged nasally with live RSV. Upon challenge, priming of mice with inactivated virus or subunit F glycoprotein induced a pattern of cytokine mRNA expression suggesting a dominant Th2-like lymphocyte response (relative increase in IL-4 mRNA expression). In contrast, challenge of mice primed with live RSV by parenteral or mucosal routes induced a Th1-like pattern of cytokine mRNA expression (relative decrease in IL-4 mRNA expression compared to IFN-.gamma. mRNA expression). Thus, the formulation and route of delivery of vaccine products can influence the pattern of cytokine expression in lung upon RSV challenge.

L15 ANSWER 47 OF 53 SCISEARCH COPYRIGHT 2003 ISI (R)
AU MEROLLA R (Reprint); PANUSKA J R; RANKIN J A; REBERT N A; TSIVITSE P T; GHNAIM H A
TI ALVEOLAR MACROPHAGES INFECTED WITH **RESPIRATORY SYNCYTIAL VIRUS** (RSV) DEMONSTRATE ALTERED **EXPRESSION** OF **CYTOKINES**
SO AMERICAN REVIEW OF RESPIRATORY DISEASE, (APR 1993) Vol. 147, No. 4, Supp. S, pp. A401.
ISSN: 0003-0805.

L15 ANSWER 48 OF 53 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC.DUPLICATE 17
AU Leopardi, R. (1); Ilonen, J.; Hurme, M.; Vainionpaa, R.; Siljander, P.; Salmi, A. A.
TI **Measles virus** modulates the **expression** of **IL**-1-beta, TNF-alpha, and MHC class II antigens in human monocytes.
SO Scandinavian Journal of Immunology, (1992) Vol. 36, No. 4, pp. 634.
Meeting Info.: XXIIIrd Meeting of the Scandinavian Society for Immunology

and the VIIIth Summer School, Oslo, Norway, May 26-31, 1992. SCAND J IMMUNOL
ISSN: 0300-9475.

L15 ANSWER 49 OF 53 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC.DUPLICATE 18
AU OKAMOTO Y; PATEL J A; TOGAWA K; SAITO I; MORO I; CHONMAITREE T; OGRA P L
TI **CYTOKINE** AND CELL ADHESION MOLECULE **EXPRESSION** DURING **RESPIRATORY SYNCYTIAL VIRUS** RSV INFECTION OF HUMAN MIDDLE EAR ME MUCOSA.
SO THIRTY-SECOND ANNUAL MEETING OF THE AMERICAN SOCIETY FOR CLINICAL NUTRITION, BALTIMORE, MARYLAND, USA, APRIL 30-MAY 2, 1992. CLIN RES. (1992) 40 (2), 379A.
CODEN: CLREAS. ISSN: 0009-9279.

L15 ANSWER 50 OF 53 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC.
AU OKAMOTO Y; PATEL J A; TOGAWA K; SAITO I; MORO I; CHONMAITREE T; OGRA P L
TI **CYTOKINE** AND CELL ADHESION MOLECULE **EXPRESSION** DURING **RESPIRATORY SYNCYTIAL VIRUS** RSV INFECTION OF HUMAN MIDDLE EAR ME MUCOSA.
SO MEETING OF THE AMERICAN PEDIATRIC SOCIETY AND THE SOCIETY FOR PEDIATRIC RESEARCH, BALTIMORE, MARYLAND, USA, MAY 4-7, 1992. PEDIATR RES. (1992) 31 (4 PART 2), 153A.
CODEN: PEREBL. ISSN: 0031-3998.

L15 ANSWER 51 OF 53 MEDLINE DUPLICATE 19
AU Zawatzky R; Wurmbaeck H; Falk W; Homfeld A
TI Endogenous interferon specifically regulates **Newcastle** disease **virus**-induced **cytokine** gene **expression** in mouse macrophages.
SO JOURNAL OF VIROLOGY, (1991 Sep) 65 (9) 4839-46.
Journal code: 0113724. ISSN: 0022-538X.
AB In macrophages from inbred mice, the magnitude of the interferon (IFN) response to Newcastle disease virus (NDV) infection is under genetic control of the If-1 locus, which carries the allele for either high (h) or low (l) IFN production. Here, we report that the activity of genes within the If-1 locus is influenced by macrophage-derived endogenous IFN. In addition to various other biological effects, we observed that endogenous IFN specifically downregulated NDV-induced IFN and interleukin 6 production. Preculture of bone marrow-derived macrophages (BMM) from BALB/c (If-1l) mice in macrophage colony-stimulating factor plus anti-IFN-beta provoked a 30- to 50-fold increase in NDV-induced cytokine production compared with induced control cultures in macrophage colony-stimulating factor alone, whereas only a 4- to 6-fold increase was observed in anti-IFN-beta-treated BMM from C57BL/6 (If-1h) mice. This resulted in nearly complete abrogation of the genetically determined difference in the response to NDV. The increase was specific for NDV and was marked by strong additional activation of IFN-alpha genes. Studies using BMM from B6.C-H28c If-1l congenic mice gave results identical to those obtained with BALB/c BMM. Addition of 20 IU of recombinant IFN-alpha 4 to anti IFN-beta-treated macrophages from B6.C-H28c mice 20 h prior to NDV infection strongly downregulated the IFN-alpha, IFN-beta, and interleukin 6 responses. The genetic difference between macrophages from If-1h and If-1l mice was thus reestablished, since the same treatment caused only weak reduction of NDV-induced cytokine gene expression in BMM from C57BL/6 mice. These data suggest that the If-1h and If-1l alleles harbor IFN-inducible genes that, following activation, specifically suppress subsequent cytokine gene expression in response to NDV.

L15 ANSWER 52 OF 53 CAPLUS COPYRIGHT 2003 ACS
AU Zawatzky, Rainer; Homfeld, Angela
TI Regulation by endogenous interferon of virus-induced cytokine gene expression in mouse macrophages
SO Pathobiology (1991), 59(4), 232-6

CODEN: PATHEF; ISSN: 1015-2008

AB In macrophages from inbred mice, the magnitude of the interferon (IFN) response to Newcastle disease virus (NDV) infection is under genetic control of the locus If-1, with C57BL/6 carrying the high-producer allele IF-1h whereas BALB/c have the low-producer allele If-1l. The IFN produced consists of 90% IFN-.beta., and there are 10-fold differences between macrophages from If-1h and If-1l mice. Interleukin-6 (IL-6) is coinduced by NDV in macrophages and seems to be under the same genetic control. Noninduced macrophages secrete low amts. of antiviral activity endogenously when cultured in the presence of the macrophage colony-stimulating factor (M-CSF). The amt. of this endogenous IFN varies between macrophages from different mouse strains. Macrophages from BALB/c secrete 5-10 times more endogenous IFN compared to C57BL/6. The antiviral activity could be identified as IFN-.beta.. Endogenous IFN specifically down-regulates NDV-induced IFN and IL-6 prodn. Preculture of BALB/c macrophages in M-CSF plus anti-IFN-.beta. to neutralize the biol. effects of the endogenous IFN provoked a 30-50-fold increase in NDV-induced cytokine prodn., resulting in a nearly complete abrogation of the genetically detd. difference, since the same treatment only caused a 6-fold increase in C57BL/6 macrophages following NDV infection. This increase in cytokine gene expression was specific for NDV and marked by a strong addnl. activation of IFN-.alpha. genes. Addn. of mouse recombinant IFN-.alpha.4 to anti-IFN-.beta. treated macrophages for 18 h prior to NDV infection down-regulated again IFN gene expression and reestablished the genetic differences between macrophages from If-1h and If-1l mice. Studies using macrophages from B6.C-H28c congenic mice, carrying the histocompatibility locus H-28 and the If-1l locus from BALB/c on a C57BL/6 background, gave identical results as with BALB/c macrophages. These data indicate that the two alleles If-1h and If-1l may harbor IFN-inducible genes responding differently to IFN and specifically suppressing subsequent IFN and IL-6 gene expression after stimulation by NDV.

L15 ANSWER 53 OF 53 CAPLUS COPYRIGHT 2003 ACS

AU D'Addario, Mario; Roulston, Anne; Wainberg, Mark A.; Hiscott, John

TI Coordinate enhancement of cytokine gene expression in human immunodeficiency virus type 1-infected promonocytic cells

SO Journal of Virology (1990), 64(12), 6080-9
CODEN: JOVIAM; ISSN: 0022-538X

AB A promonocytic cell model was used to investigate cytokine gene transcription in U937 and U9-IIIB cells chronically infected with human immunodeficiency virus type 1 (HIV-1). The prodn. of interferon (.alpha.-1 interferon [IFN-.alpha.1], IFN-.alpha.2, and IFN-.beta.) interleukin (interleukin 1.alpha. [IL-1.alpha.], IL-1.beta., and IL-6), and tumor necrosis factor .alpha. (TNF-.alpha.) was characterized by quant. polymerase chain reaction mRNA phenotyping in U937 and U9-IIIB cells following coinfection with Sendai paramyxovirus or stimulation with lipopolysaccharide (LPS). Chronic HIV-1 infection of U9-IIIB cells resulted in a low constitutive level of transcription of TNF and IL-1 genes but not IFN genes; however, when the cells were coinfected with Sendai virus, 10- to 20-fold higher levels of IFN-.beta., IL-1.beta., IL-6, and TNF-.alpha. mRNA were obsd. in U9-IIIB cells than in similarly induced U937 cells. The enhanced levels of cytokine RNA in virus-infected U9-IIIB cells were also accompanied by higher levels of IFN antiviral activity and TNF secretion than in U937 cells. Transcript levels for IFN-.alpha.1 and IFN-.alpha.2 were equivalently induced in virus-infected U937 and U9-IIIB cells, indicating that a generalized depression of cytokine gene expression did not occur as a consequence of HIV-1 infection. When LPS was used as an inducer, a distinct pattern of cytokine expression was detected in U9-IIIB cells. TNF-.alpha. and IL-1.beta. but not IFN-.alpha. or IFN-.beta. transcripts were induced by LPS. These results suggest that HIV-1 infection of promonocytic cells may prime or sensitize cells such that subsequent antigenic challenge leads to coordinate enhancement of cytokine gene expression.